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Syncytin is a captive retroviral envelope protein involved in human placental morphogenesis.

MI S, Lee X, Li X, Veldman GM, Finnerty H, Racie L, LaVallie E, T: XY, Edouard P, Howes S, Keith JC Jr, McCoy JM.

Genetics Institute, Inc., Cambridge, Massachusetts 02140, USA.

Many mammalian viruses have acquired genes from their hosts during the evolution. The rationale for these acquisitions is usually quite clear: the captured genes are subverted to provide a selective advantage to the virus. Here we describe the opposite situation, where a viral gene has been sequestered to serve an important function in the physiology of a mammal host. This gene, encoding a protein that we have called syncytin, is the envelope gene of a recently identified human endogenous defective retrovirus, HERV-W. We find that the major sites of syncytin expression are placental syncytiotrophoblasts, multinucleated cells that originate from fetal trophoblasts. We show that expression of recombinant syncytin in a wide variety of cell types induces the formation of giant syncytia, and that fusion of a human trophoblastic cell line expressing endogenous syncytin can be inhibited by an anti-syncytin antiserum. Our data indicate that syncytin mediates placental cytotrophoblast fusion in vivo, and thus may be important in human placental morphogenesis.

PMID: 10693809 [PubMed - indexed for MEDLINE]

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FULL TEXT ARTICLE**Downregulation of placental syncytin expression and abnormal protein localization in pre-eclampsia.****Lee X, Keith JC Jr, Stumm N, Moutsatsos I, McCoy JM, Crum CP, Genest D, Chin D, Ehrenfels C, Pijnenborg R, van Assche FA, Mi S.**

Wyeth/Genetics Institute, One Burt Road, Andover, MA 01810, USA.

Development of placentation and successful pregnancy depend on coordinated interactions between the maternal decidua and myometrium, and the invasive properties of the fetal trophoblast. Syncytin, a protein encoded by the envelope gene of a recently identified human endogenous defective retrovirus, HERV-W, is highly expressed in placental tissue. Previously, we have shown that the major site of syncytin expression is the placental syncytiotrophoblast, a fused multinuclear syncytium originating from cytotrophoblast cells. Here we present the first evidence that in pre-eclampsia, syncytin gene expression levels are dramatically reduced. Additionally, immunohistochemical examination of normal placentae and placentae from women with pre-eclampsia reveals that the syncytin protein in placental tissue from women with pre-eclampsia is localized improperly to the apical syncytiotrophoblast microvillous membrane as opposed to its normal location on the basal syncytiotrophoblast cytoplasmic membrane. Previous results suggest that syncytin may mediate placental cytotrophoblast fusion in vivo and may play an important role in human placental morphogenesis. The present study suggests that altered expression of the syncytin gene, and altered cellular location of its protein product, may contribute to the aetiology of pre-eclampsia. Copyright 2001 Harcourt Publishers Ltd.

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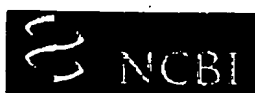
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FULL-TEXT ARTICLE

Syncytin, a novel human endogenous retroviral gene in human placenta: evidence for its dysregulation in preeclampsia and HELLP syndrome.

Knerr I, Beinder E, Rascher W.

Department of Pediatrics, University of Erlangen-Nuremberg, Germany.

OBJECTIVE: A novel human endogenous retroviral element, designated syncytin, has been suggested as a contributor to normal placental architecture especially in the fusion processes of cytotrophoblasts to syncytiotrophoblast. We tested the hypothesis of whether the gene expression of syncytin may be altered in cases with placental dysfunction such as preeclampsia or HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome. **STUDY DESIGN:** We included 30 women with normal pregnancies, 16 with preeclampsia, and 6 with HELLP syndrome. After delivery, messenger ribonucleic acids (mRNA) of syncytin, glyceraldehyde-3-phosphate dehydrogenase and beta-actin were analyzed in placental villi with use of quantitative real-time polymerase chain reaction. **RESULTS:** In placental villi, syncytin mRNA/beta-actin mRNA and syncytin mRNA/glyceraldehyde-3-phosphate dehydrogenase mRNA ratios were lower in patients with preeclampsia ($P < .05$) or HELLP syndrome than in healthy control subjects. **CONCLUSION:** A reduced placental expression of syncytin may contribute to altered cell fusion processes in placentogenesis and disturbed placental function in hypertensive disorders of pregnancy.

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Hypoxia alters expression and function of syncytin and its receptor during trophoblast cell fusion of human placental BeWo cells: implications for impaired trophoblast syncytialisation in pre-eclampsia.

Kudo Y, Boyd CA, Sargent IL, Redman CW.

Department of Human Anatomy and Genetics, University of Oxford, Sou Parks Road, OX1 3QX, Oxford, UK. yoshkudo@hiroshima-u.ac.jp

The fundamental process of placental trophoblast cell fusion (syncytiotrophoblast formation or syncytialisation) which is a characteristic of this tissue is poorly understood. Pre-eclampsia is associated with placental hypoxia and suppressed syncytiotrophoblast formation. We therefore have studied the effect of low-oxygen tensions on the rate of cell fusion, relative abundance of mRNAs encoding syncytin and its receptor, amino acid transport system B(0), which are thought to be involved in trophoblast cell fusion (as well as the activity of amino acid transport through this system) in a cell model of syncytialisation (BeWo cells following forskolin treatment). Forskolin-induced cell fusion (determined by a quantitative flow cytometry assay) was reversibly suppressed in 2% oxygen compared to 20% oxygen. This was associated with suppressed secretion of human chorionic gonadotropin. Forskolin stimulated relatively less syncytin mRNA (determined by reverse transcription-polymerase chain reaction) in 2% than in 20% oxygen and there was no stimulation after 48 h in 2% oxygen. There was a spontaneous, time-dependent increase of amino acid transporter B(0) mRNA in vehicle, which was suppressed by 2% oxygen and by forskolin treatment in 20% oxygen. Forskolin-induced changes in amino acid transport system B(0) function were not seen in cells cultured for 48 h in 2% oxygen. These observations suggest that under conditions of low ambient oxygen, dysregulation of expression of syncytin and of its receptor may suppress the normal process of cell fusion necessary for syncytiotrophoblast formation and contributes to syncytiotrophoblast abnormalities characteristic of pre-eclampsia.

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